*J. Agric. Sci. Mansoura Univ., 29(5): 2253 - 2272, 2004*

# PERVALENCE OF MASTITIS PATHOGENS AND HISTOPATHOLOGICAL CHANGES OF THE MAMMARY GLAND IN THE EGYPTIAN BUFFALO

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## ABSTRACT

Physical examination combined with bacterial isolation and histological investigation were used to assess the mammary gland health condition in Egyptian buffalo. Out of four hundreds lactating buffaloes examined physically. Examination of ٥٠٨ milk samples collected from 127 buffaloes for bacterial isolates was carried out. The percentage of abnormal udders reached ٧,٧٥ %. The most observed abnormalities were supernumerary teats, blind quarters, extrateat orifice, stepped udders and atrophied quarters. ٨٢ % of the examined samples had high total bacterial counts (TBC), indicating that the sanitation conditions of the buffaloes' environment is not appropriate. Moreover, the most prevalent mastitis pathogens were Staphylococcal isolates (٤٨,١٨ %), *Streptococcus dysagalactiae* (٢٣,٦٩ %), Bacillus (٩,٢ %), and Coliform (٥,٦ %).

When the mammary tissue submitted to detailed histological investigation, severe deterioration of secretory tissue which is mostly replaced by connective tissue stroma accompanied with inflammatory and fibroblastic cell infiltration was found. Moreover, corpora amylacea was found in the lumen of some of the degenerated acini. These histological alterations were accompanied with the occurrence of multiple infection of the mammary gland. it was concluded that great deal of attention is required to diagnose, segregate and properly treat the infected buffaloes. On the other hand, early culling of the buffaloes with distorted mammary gland should greatly decrease the chance of the vulnerable tissue for pathogenic infection.

**Keywords:** udder, buffalo, histopathology, bacteriology, mastitis.

## INTRODUCTION

Pathogenic infection of the mammary gland can manifest itself on a variety of forms depending upon the causal agent such as animal factors (breed, udder morphology, stage and order of lactation), environmental factors (season), and farm management practices such as sanitation, (Thirunvukkarasu and Prabaharan, ١٩٩٨ b). When the pathogenic organisms passes through the cistern and ducts of a susceptible udder, multiplication of the organisms occur, but may remain latent for varying periods of time with no detectable signs. At this time, the presence of the organisms can not be detectable by the routinely used mastitis screening tests, even though these organisms still capable of causing clinical disease reviewed by (Schalm *et al.*, 1971). When the organisms pass through and penetrate the tissue of the mammary gland, a " flare up " followed by an inflammatory reaction occur. This inflammation is characterized by influx of white blood cells into the mammary tissue followed by increasing mammary epithelial cells as a result of mastitis. This leads to the reduced synthesis and secretion of milk components synthesized de novo; thus the resulting

Assessment of damaged secretory tissue of the mammary gland affected by infection through histological examination revealed less synthesizing and secretory activity as evidenced by more interalveolar stroma and involuting alveolar luminal space (Nickerson and Heald, 1981 and Sordillo and Nickerson, 1988). Thus infective pathogens may have deleterious effects not only in the current lactation, but also may be extended to future milk yield if the causative effect is not appropriately dealt with either through treatment or culling.

Little information are a available concerning the prevalence of mastitis pathogens in the buffaloes, especially in Egypt, and the effect of such an infection on the functional activity of the udder tissues. Therefore, the aims of the present work were, firstly to examine the udders physically and classify it as healthy or non-healthy based on the presence of signs of udder distortion, secondly, to determine the prevalence of bacteriological agents that causes mastitis. The third goal was to study the histological changes resulting from the infection with mastitis pathogens on the mammary tissue.

## MATERIALS AND METHODS

**A. Experimental herd and physical examination:**

A buffalo herd of 400 females at Mehallet-Moussa experimental station located in the northern Delta were examined physically for either having healthy udders or bearing signs of udder distortion. These buffaloes were under routine management and feeding according to APRI (1997). The ages of this herd ranged from 3 to 19 years. No available data regarding previous history of mastitis infection or treatment of this buffalo herd.

**B. Sampling and bacteriological examination:**

Milk samples were collected from 127 buffaloes (508 quarters). The parity distribution of the milking buffaloes is shown in Table (1). Each milk sample was cultured and the total number of bacteria was determined according to the standard methods (Houghtby *et al.,* 1992). Detection and enumeration of specific organisms were performed by serial dilution of milk samples that were spread-plated onto MacConkey agar for presumptive Gram-negative (Coliform counts), modified Edward's medium for presumptive *Streptococcus spp.* (agalactiae, dysagalactiae, and uberis), Staph-110 media for presumptive *Staphylococcus spp.*, Bird Parker agar with Tellurite for presumptive corynebacterium, and finally blood agar for *Bacillus spp.* All plates were incubated at 37°C±2°C, then examined 24 hr and 48 hr later. These selective and differential media were chosen for the isolation and identification of mastitis pathogens according to Collins and Patricia (1979). The results were expressed as cfu (colony forming unit)/ ml.

**C. Biopsy and histological examination:**

A sample of five buffaloes proved positive for mastitis pathogens and apparently having distorted udders, in addition to one buffalo free of mastitis pathogens with apparently healthy udder were biopsied. Mammary tissue was obtained from each buffalo by surgical biopsy as described by Mellenberger, *et al.* (1973). Each sample weighed 10-15 g and the tissue was immediately fixed in Bouin's solution for 24-48 hr., then transferred to be preserved in 20 % ethylalcohol. Processes of dehydration, clearing,

impregnation and staining with Hemotoxylin and Eosin were accomplished according to clark (1981). The histological examination was done by means of research Microscopy.

**Table (I): Distribution of the parity of the buffalo herd subjected to bacteriological examination.**

| Parity | Percentage of the herd |
|---|---|
| 1st | 9 |
| 2nd | 12 |
| 3rd | 17 |
| 4th | 9 |
| 5th | 7 |
| 6th | 9 |
| 7th | 12 |
| 8th | 9 |
| 9th | 9 |
| 10th or after | 7 |
| Total | 100 |

## RESULTS AND DISCUSION

**1. Physical examination of the mammary gland:**

Four hundreds buffaloes were physically examined for apparently having healthy udders or carrying signs of udder distortion. The percentage of apparently healthy udders reached 92.25 % (Table 2). The udders classified as on-healthy included teat paralysis, stepped udders (distorted and unorganized teats), blind quarters, teats with two orifices, teat keratinization, attached teats, presence of large nodules in the teats, supernumerary teats, in addition to an oedematous udder (Table 2 and Plates 1, 2, 3 and 4). Some of the visible and palpatory changes in the mammary gland noticed in the present study could be congenital and represent a predisposing factor for mastitis such as blind quarters, supernumerary teats, stepped udders (Bowley and Weaver, 1999).

**Table (2): The percentage of various kinds of abnormalities observed through physical examination of the udder of experimented buffaloes.**

| Abnormality | Percentage |
|---|---|
| Necrotic | 1.5 |
| Stepped udders | 2.25 |
| Blind quarters | 0.25 |
| An extra teat orifice | 0.25 |
| Attached teats | 0.25 |
| Presence of large nodes | 1.75 |
| Supernumerary teats | 1.25 |
| Atrophied udders | 0.25 |
| Total | 7.75 |


Plate 1. Udder with nodular teat (N) and atrophied quarters (A)

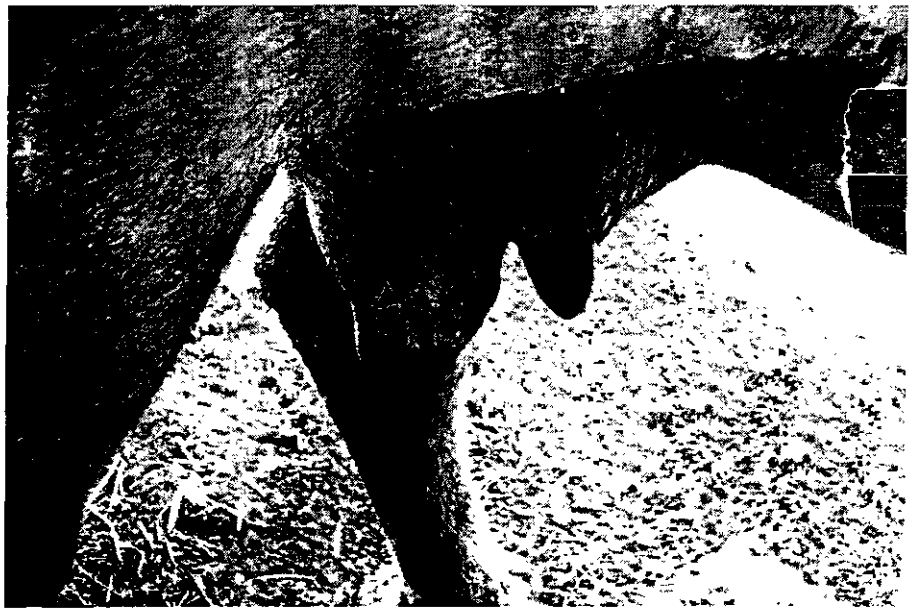

Plate 2. Udder with two attached quarters

Plate 3. Atrophied udder with supernumerary teat.

Plate 4. Unorganized udder with two attached quarters.

**Bacteriological Findings:**

Table (3) summarizes the percentage of the infected quarters in the examined buffaloes. Overall 82 % of the quarters were found positive concerning the total bacterial count (TBC). TBC is known to serve as a rough guage of herd health, farm sanitation efficiency and proper handling of milk (Hayes, *et al.*, 2001). As shown in Table (3) out of 249 quarters found positive for total bacterial count, 98 quarters (39.65 %) were infected with contagious bacteria. This group of pathogens included *Streptococcus agalactiae* (4 %), *Staphylococcus auries* (2.4 %), *Streptococcus dysagalactiae* (23.69 %) and corynebacteria (12.85 %). However, the percentage of quarters infected with bacterial pathogens that considered not contagious in nature (transmitted through animal's environment) reached 60.6 % including Staphylococcus isolates (45.78 %), Coliform (5.62 %), and Bacillus (9.24 %) (Table 3). The most prevalent isolates was the Staphylococci ( 48.18%) including *Staphylococcus auries* (2.4 %) and other Staphylococcus isolates (45.8 %); followed by *Streptococcus dysagalactiae* (23.69 %).

**Table (3): Prevalence of mastitis pathogens in buffalo's milk samples examined individually.**

| Trait | N | % of infected quarters |
|---|---|---|
| Total bacterial count (TBC)* | 249 | 100 |
| **a. Contagious bacteria:** | 98 | 39.35 |
| *Strept. Agalactiae* ** | 1 | 0.4 |
| *Sterpt. Dysagalactiae* | 6 | 23.69 |
| *Staph. Auries* | 59 | 2.4 |
| Corynebacteria | 32 | 12.85 |
| **b. Environmental bacteria:** | 151 | |
| *Strept. Uberis* | 0 | 0 0 |
| *Staph. (other)* | 114 | 45 8 |
| Coliform | 14 | 5.6 |
| Bacillus | 23 | 9 2 |

**N = Number of infected quarters.**

*** Percentage of quarters with high TBC is calculated as number of quarters with h.gh total bacterial count to the total number of the examined quarters.**

****Percentage of quarters considered positive for a specific pathogen is calculated as number of quarters showed higher than the maximum number allowed for this patnogen to the total number of quarters with high TBC.**

**Histological Findings:**

The histological observation of the mammary gland tissue taken from buffaloes bearing signs of udder distortions and infected with mastitis pathogens showed various degrees of inflammatory changes and leucocyte infiltration in addition to severe destruction of the secretory tissue. Some of the mammary lobules showed oedema with inflammatory cell infiltration in-between the degenerated acini which had desquamated cells in the acinar lumen (Plate 5). In other lobules, the acini were atrophied and surrounded by inflammatory acini were atrophied and surrounded by inflammatory cell infiltration and fibroblastic proliferation (Plate6)

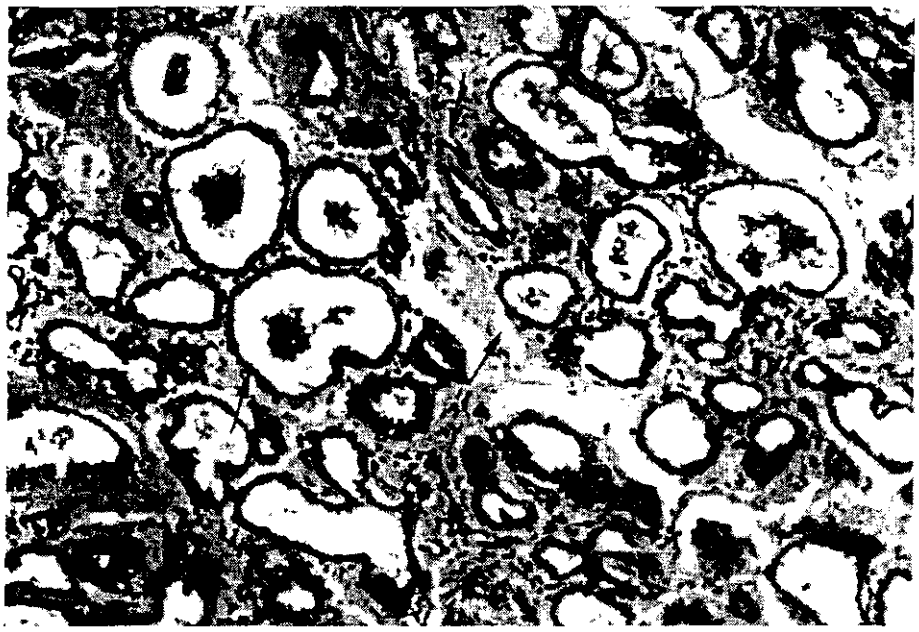

Plate 5. A degenerated acini of the mamary gland infiltrated by inflammatory cells and associated with desquamated cells in the acinar lumen. X=40. H&E

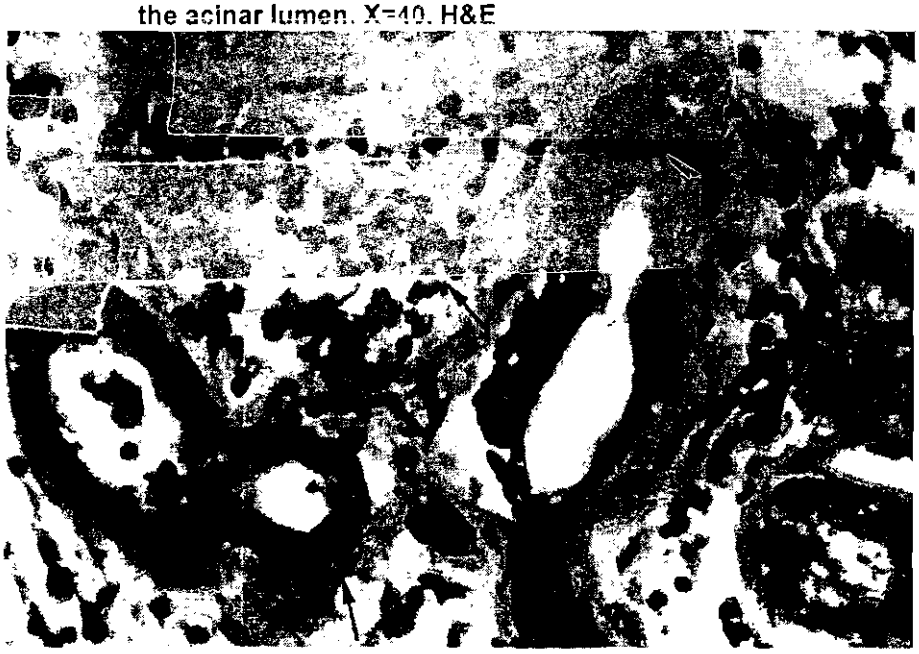

Plate 6. The mammary gland showing atrophied acini infiltrated by inflammatory cells. X = 160. H&E

Newly formed capillaries and fibroblastic cells were observed in-between the atrophied acini (Plate 7). There was focal extravasated red blood cells and oedema in-between the mammary lobules (Plate 8). The histopathological findings was the result of the infection that lead to chronic mastitis (Heidrich and Renk, 1967). In another case infected with Staphylococcus, the mammary tissue showed mononuclear leucocytic inflammatory cells infiltration and fibroblastic proliferation in-between the nonfunctioning atrophied acini. This was associated with hyperplasia in the lining epithelium of the interlobular lactiferous ducts forming polyps like projections protruded in the ductal lumen (Plate 9). There was a diffuse proliferation of fibroblast cells over the lobules with appearance of desquamated cells and eosinophilic exudates in the lumen of the cystically dilated interlobular lactiferous ducts (Plate 10). As a result of Staphylococci and Coliform infection in 3 quarters of the udder, the mammary tissue showed a massive number of extravasated red blood cells in the acini showing necrobiotic changes (Plate 11) as well as in the lumen of the interlobular lactiferous ducts (Plate 12). Moreover interlobular connective tissue stroma showed a massive number of extravasated red blood cells (Plate 13). There was severe dilation in the blood vessels associated with inflammatory cells infiltration and fibroblastic proliferation in connective tissue stroma of the atrophied acini. The lumen of the intralobular lactiferous ducts was occluded by mass of necrosed desquamated tissue and blood cells (Plate 14). Diffuse proliferation of fibroblasts were noticed in-between the atrophied acini in the mammary lobules (Plate 15). There was also homogenous oesinophilic lamellated mass (corpora amylacea) in the lumen of some degenerated acini (Plate 16). Degenerative changes associated with interstitial basophilic corpora amylacea in-between the lobules was also observed in the mammary tissue infected with *Streptococcus agalactiae* and Bacillus (Plate 17). Focal lymphoid cells aggregations were noticed in the mammary lobules (Plate 18) accompanied by periductal leucocytic inflammatory cells infiltration surrounded by the degenerated interlobular lactiferous ducts (Plate 19).

The ultimate goal of the current study was the detection of clinical and subclinical mastitis using physical examination in combination with bacteriological assessment and histological investigation. Physical examination illustrates teat lesions, and other miscellaneous conditions that predispose the udder vulnerable to infection with mastitis pathogens (Thirunvukkarasu and Prabaharan, 1998a). Teats are known to be vulnerable to injuries, eczema and other physical influences. No accurate statistics available concerning the abnormalities of the mammary gland in the adult buffaloes, may be due to early culling of the heifers that have any visible changes in their udder before it reach the productive stage. The visible changes in the udder of the investigated herd reached 7.75 % (Table 2). These changes could be classified into congenital and non-congenital (acquired) alterations. The congenital udder abnormalities included blind quarters, on which complete absence of the teat canal may be found. Also, supernumerary teats, in that case, the extra teat could be found attached to the base or the side of one of the main teats where they can interfere with

milking. The extra teats in their typical shape are shorter than normal and have thinner walls. Moreover, in stepped udders (distorted or unorganized quarters), the use of milking machines may be hindered or completely prevented due to unequal or unorganized quarters. (Bowley and Weaver, 1999).

The alterations in the teats, orifices or the main duct and the cistern could also be held responsible for difficult hand or machine milking. This could be acquired in later life through diseased udder or by the milking methods. The acquired alterations include necrotic teats, atrophied quarters in addition to quarters having large, hard nodules. The latter one is known to be chronic intramammary staphylococci abscesses (Heidrich and Renk, 1967). Atrophied quarters and necrotic teats are known to result from chronic infection with mastitis pathogens that are not detected or treated. Since the present study was conducted to examine the prevalence of bacteriological mastitis pathogens in Egyptian buffalo, milk of 127 lactating buffaloes (508 individual quarters) was examined for both contagious and environmental (non-contagious) pathogens. Eighty two percent of the examined quarters showed high total bacterial count (Table 3). Hayes, *et al.* (2001), indicated that the wide-spread bacterial contamination could be a consequence of multiple sources as mastitic buffaloes, dirty udders and poorly cleaned milking equipments. This conclusion was also emphasized by the results in Table (3) showing that 61 % of the quarters was influenced by environmental bacteria and 39% of samples infected with contagious types. Reneau (1986) showed that management is the major factor controlling the incidence of intramammary infection in lactating cows. In the present study, the most prevalent isolates was Staphylococcus (45.18 %) including aureus (2.4 %) and others (45.7 %), followed by *Streptococcus dysagalactiae* (23.7 %), corynebacteria (12.9 %) and Bacillus (9.2 %) (Table 3). These results were in close agreement with the results of Bansal *et al.* (1995) in cows and buffaloes and Shalaby and Mohamed (2000) in Egyptian buffaloes. The influence of infection by different bacterial isolates varies depending upon the kind of pathogen. Staphylococci has been recognized as a significant herd problem known to lead to staphylococcal mastitis. This disease in its commonest form is thought to be chronic with some mild changes in milk quantity and quality (Nickerson and Heald, 1981). However, *Streptococcus dysagalactiae* are not contagious, infection with S. *dysagalactiae* may be temporary, mild and infrequent. Even though corynebacteria are often found in freshly-drawn milk. They are rarely associated with clinical mastitis and the infection may come via the teat canal. Moreover, the wide-spread coliform bacteria, the infection comes only through direct contact of the teats with manure or soil heavily contaminated with fecal matter and the organism may produce mild clinical mastitis (Schalm, *et al.*, 1971). *Streptococcus uberis* was not isolated from any of the samples. This result may indicate natural eradication of this isolate or its vectors from the surrounding environment. Fox, *et al.* (1995) showed that management system and climatic conditions might influence the incidence of intramammary infections.

The normal mammary gland secreting tissue which is lined with simple epithelium (Plate 20), is characterized by numerous secreting acini and ducts in the mammary lobules. Infection of the mammary tissue with *Streptococcus dysagalactiae* resulted in oedema with inflammatory cell infiltration between the degenerated acini which had desquamented cells in the acinar lumen (Plate 5), in other lobules, the acini were atrophied and surrounded by inflammatory cell infiltration and fibroplastic proliferation (Plate 6). In addition to newly formed capillaries and fibroblastic cell proliferation (Plate 7). There was also extravasated red blood cells and oedema in-between the mammary lobules (Plate 8).

Infection with Staphylococcus isolates showed increased mononuclear leucocytic inflammatory cells infiltration and fibroblastic proliferation were noticed in-between the non-functioning atrophied acini in association with hyperplasia in the lining epithelium of the intralobular lactiferous ducts forming polyps like projections protruded in the ductal lumen (Plate 20). There was also diffuse proliferation of fibroblasts allover the lobules with appearance of desquamated cells and eosinophilic exudates in the lumen of the cystically dilated interlobular lactiferous ducts (Plate 10). In response to multiple infection with *Streptococcus agalactiae,* Coliform, Bacillus and Staphylococcus to the mammary tissue, massive number of extravasated red blood cells were observed in the lumen of the acini which showed necrobioic change (Plate 11), as well as in the lumen of the intralobular lactiferous ducts (plate 12), and in focal manner at the interlobular connective tissue stroma (Plate 13). There was severe dilation in the blood vessels associated with inflammatory cell infiltration and fibroblastic proliferation in connective tissue stroma between the atrophied acini (Plate 21). The lumen of the intralobular lactiferous duct was occluded by mass of necrosed desquamented tissue and blood cells (Plate 14). Diffuse proliferation of fibroblasts were noticed in-between the atrophied acini in the mammary lobules (Plate 13). There was also homogenous eosinophilic lamellated mass (corpora amylacea, in the lumen of some degenerated acini (Plate 16). Corpora amylacea arises during cysts development in the course of catarrhal inflammation of the udder. They are usually palpable as firm nodules under the movable skin, but sometimes they bulge to the surface as dome-shaped swellings (Heidrich and Renk, 1967). The severe deterioration of the mammary tissue in this case may be due to chronic and recurrent infection with multiple mastitis pathogen as was found in this study. Another case of mammary tissue multiple infection was also observed, in which, infection with Staphylococcus isolates. Streptococcus isolates, and Bacillus resulted in degeneration of the epithelial cells lining the acini of the mammary lobules (Plate 20), associated with interstitial lamellated basophilic corpora amylacea in-between the lobules (Plate 10). Mononuclear leucocytic inflammatory cells infiltration and few fibroplastic cells proliferation were observed in diffuse manner in the interlobular as well as intracinar connective tissue stroma (Plate 11). Focal lymphoid cells aggregations were noticed in the mammary lobules (Plate 13), accompanied by periductal leucocytic inflammatory cells infiltration surrounded by the degenerated interlobular lactiferous duct (Plate 21).

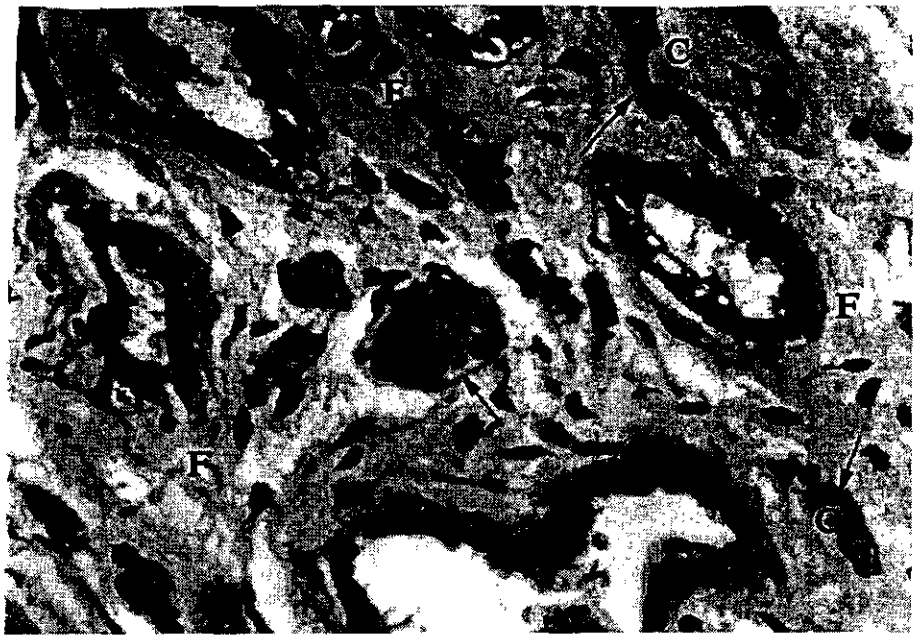


Plate 7. The atrophied acini of the mammary gland penetrated by newly formed capillaries (C ) and fibrosis (F). X = 160. H&E

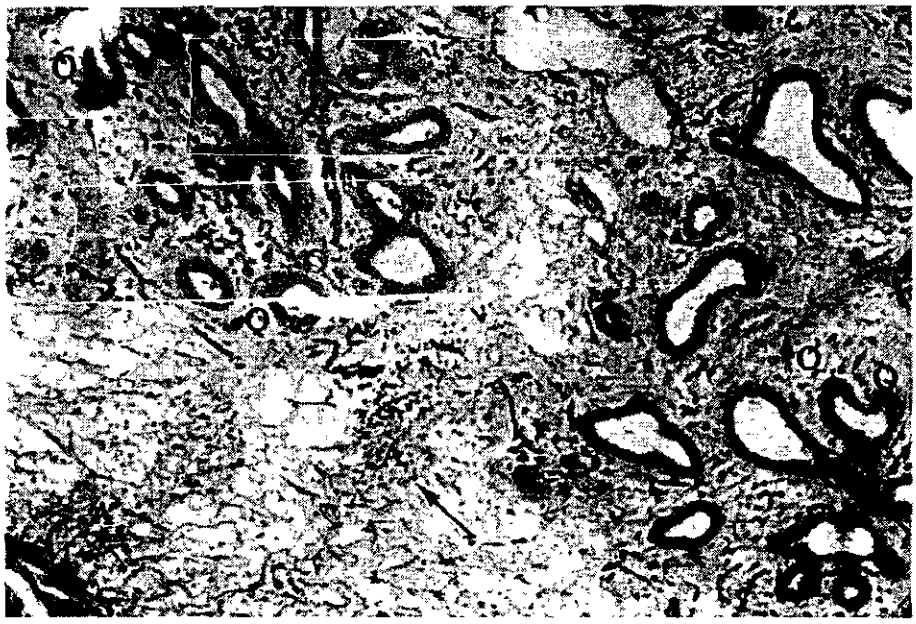


Plate 8. The mammary gland showing focal extravasation of red blood cells (A) and oedema penetrating the lobules (O). X= 40. H&E

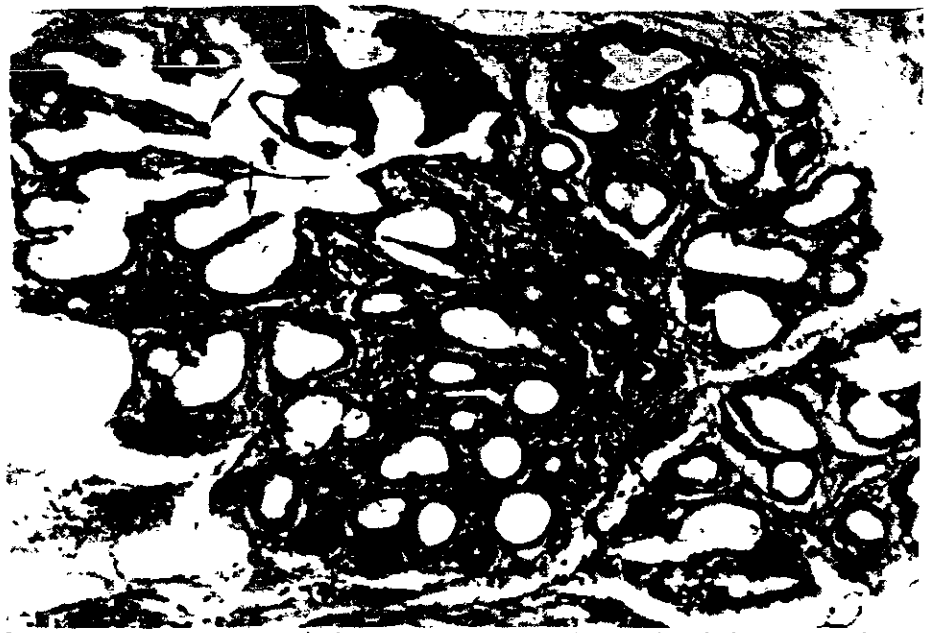

Plate 9. The stroma of the mammary gland showing infiltration of the mononuclear leucocytic inflammatory cells (M), proliferated fibroblast (P), atrophy of the acini (A) and hyperplasia of the interlobular lactiferous duct (H). X= 40. H&E

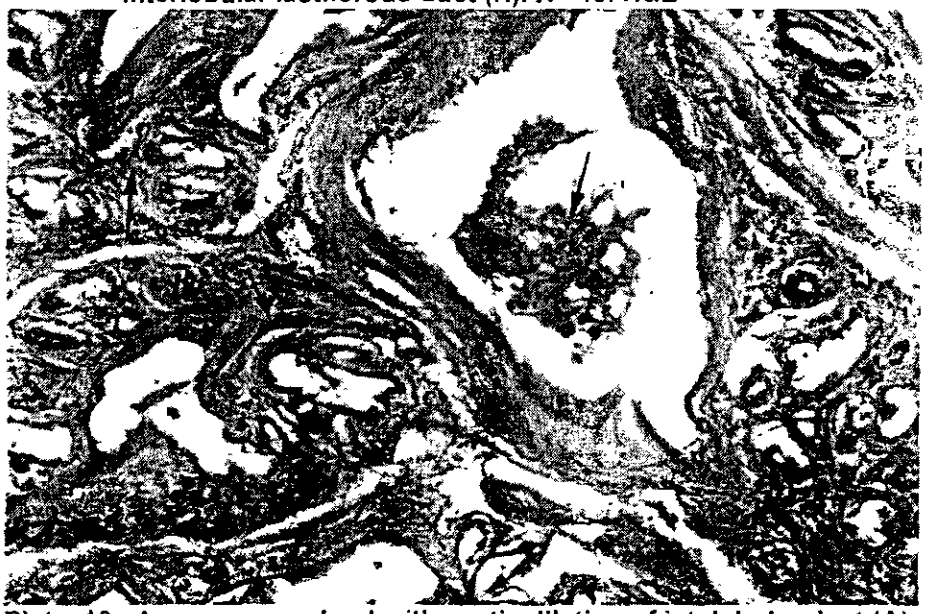

Plate 10. A mammary gland with cystic dilation of intelobular duct (A), desquamated cells in the duct of lumen (D)and an atrophied non-functioning acini(A) penetrated by fibrous tissue proliferation(F). X=40. H&E

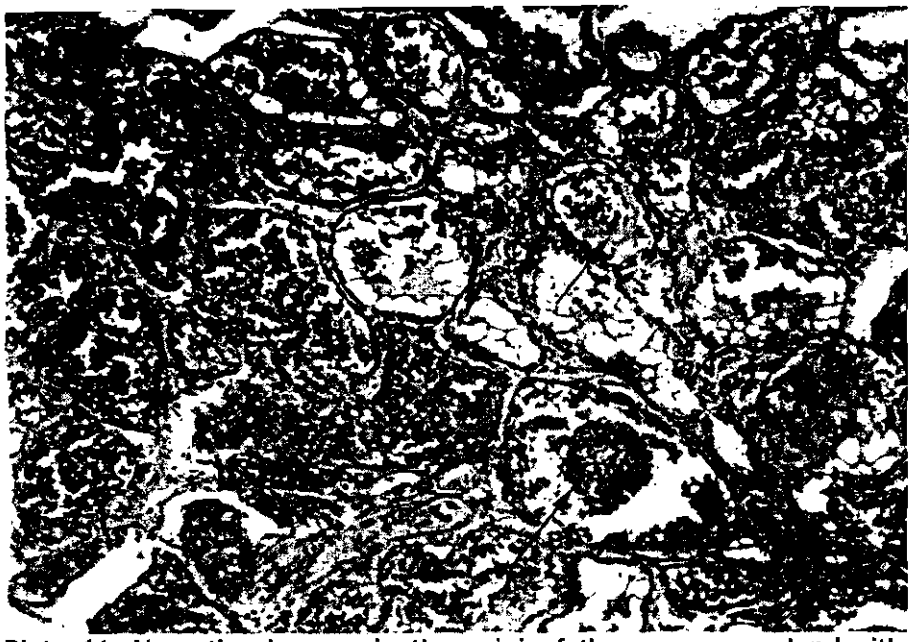

Plate 11. Necrotic changes in the acini of the mammary gland with massive number of extravasated red blood cells impacted the acinar lumen. X=40. H&E

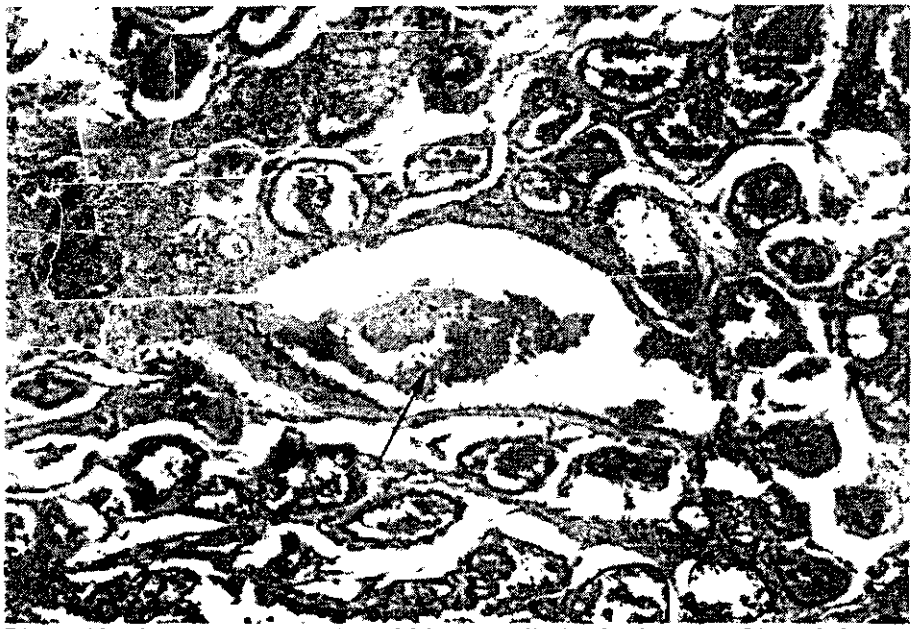

Plate 12. An extravasated red blood cells in the lumen of intralobular lactiferous duct of the gland with desquamation of the ductal lining epithelium. X=40. H&E

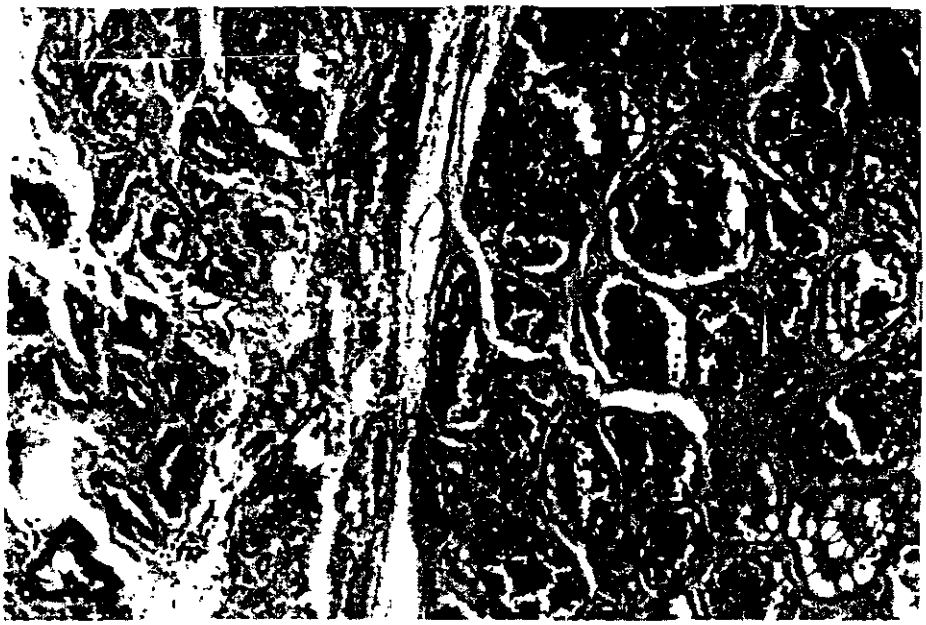

Plate 13. the mammary gland with massive number of extravasated red blood cells in the interlobular stroma. X=40. H&E

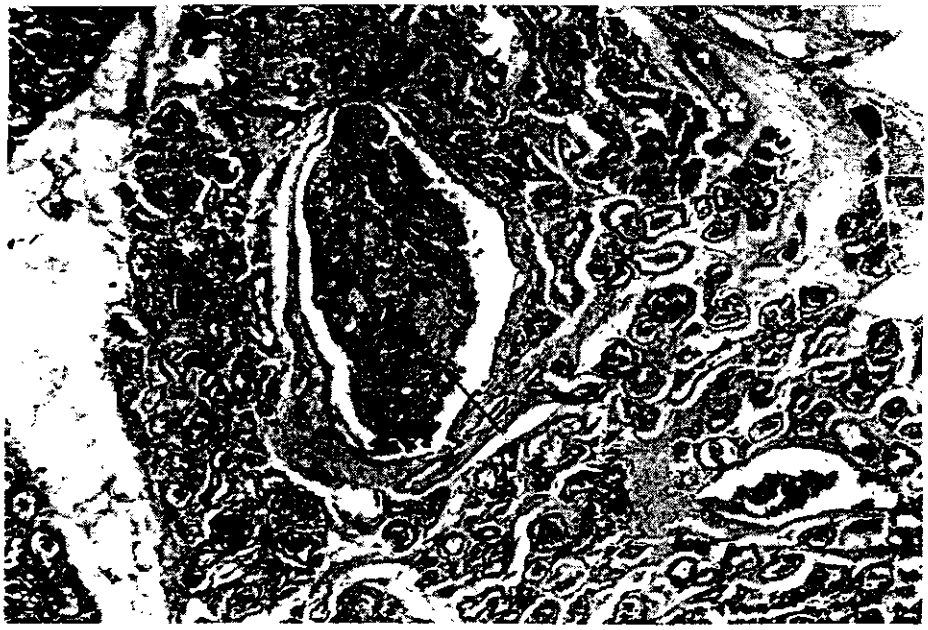

Plate 14. Necrosed desquamated tissue mass with blood cells in the lumen of the intralobular lactiferous duct of the mammary gland. X=16. H&E

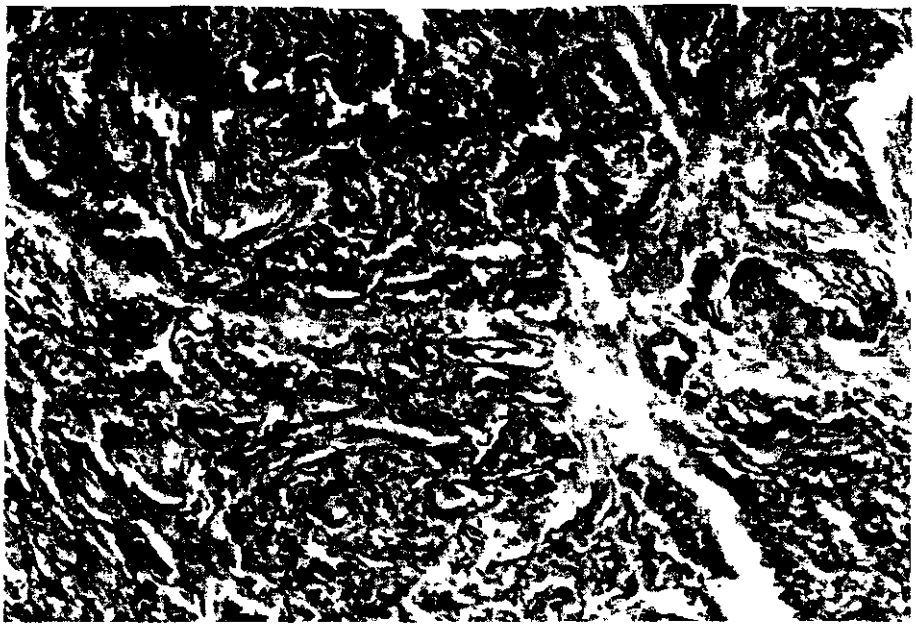

Plate 15. Diffuse fibrosis penetrating the atrophied acini in the mammary lobule. X = 40. H&E

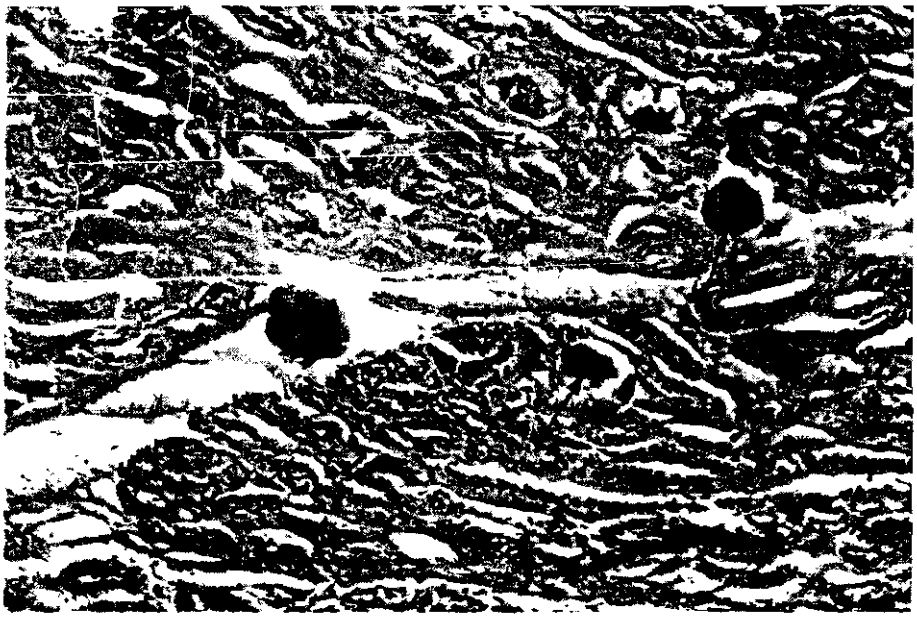

Plate 16. Corpora amylacea in the lumen of the atrophied acini of the mammary lobule. X = 40 H&E

Plate 17. The interstitial corpora amylacea of the mammary gland. X=160. H&E

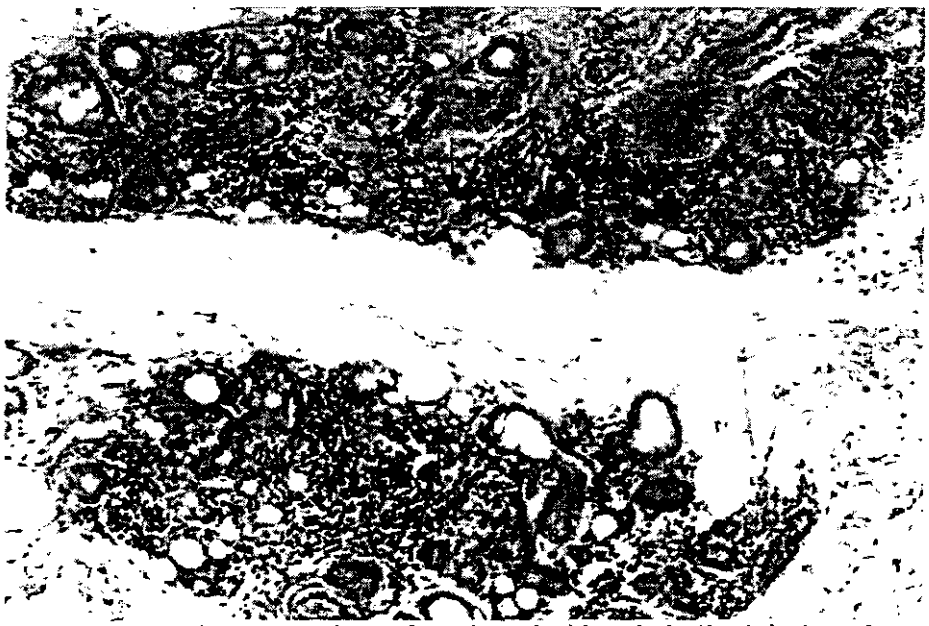

Plate 18. Focal aggregation of the lymphoid cells in the lobules of the mammary gland. X = 40. H&E

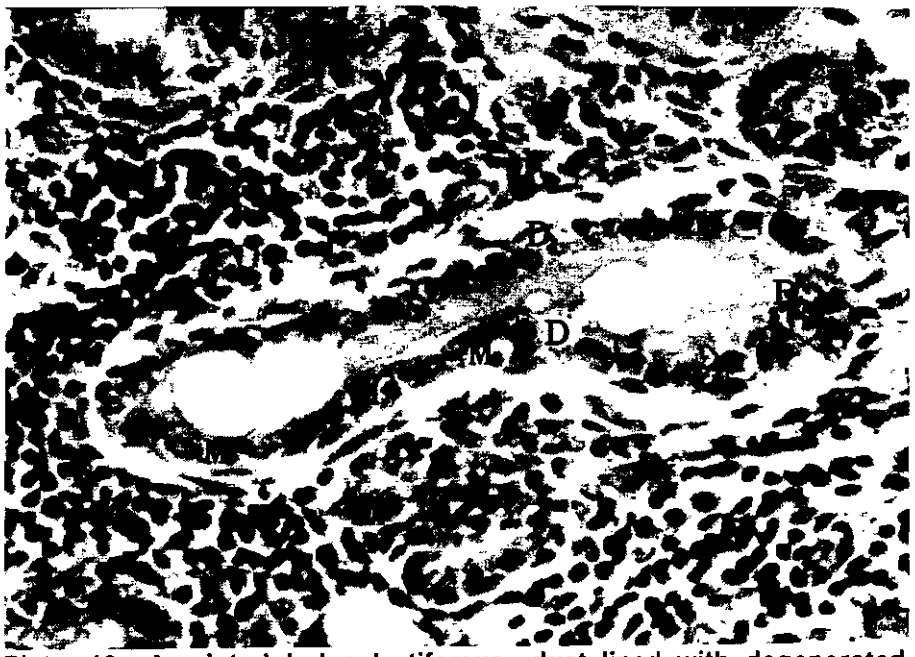


Plate 19. An interlobular lactiferous duct lined with degenerated epithelium (D) surrounded by mononuclear leucocytic inflammatory cells (M). X = 160. H&E

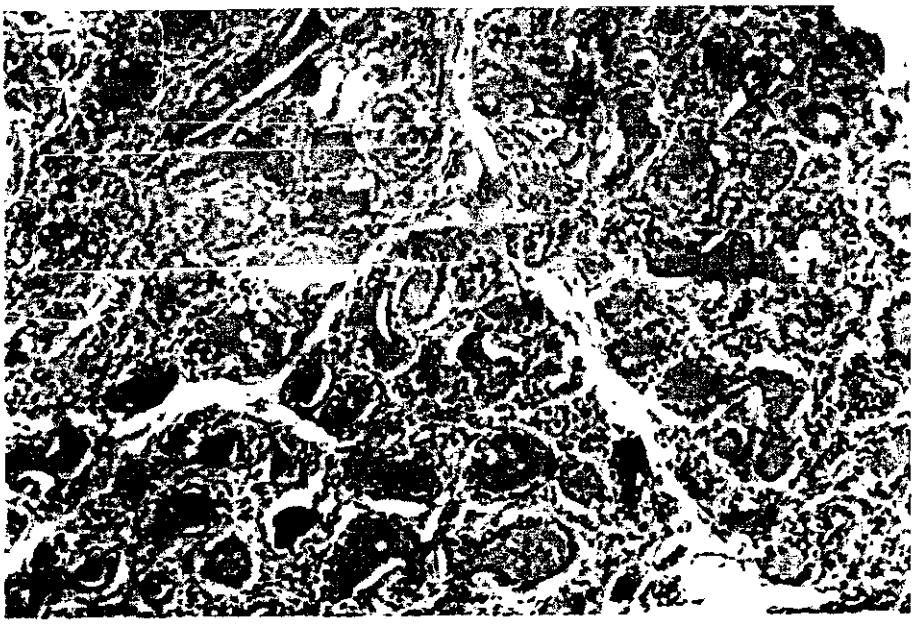

Plate 20. The normal histological secreting acini and ducts in the mammary gland lobule. X=40. H&E

**Plate 21. Dilation of the interlobular blood vessels (B) with inflammatory cells infiltration penetrating the atrophied acini (A) in the mammary gland lobule. X=40. H&E**

The current histological results agrees with the results obtained by many investigators (Heald, 1979; Nickerson and Heald, 1981; Trinidad, *et al.*, 1990; Zank and Schlatterer, 1998 and Sordillo and Nickerson, 1988) indicating that lactating animals infected with Staphylococcal and Streptococcus isolates resulted in more interalveolar stroma and involuting alveolar epithelium and less alveolar luminal space, in addition to replacement of secretory tissue with non-secretory tissue. Moreover, inflammatory changes and heavy leucocyte infiltration observed in the infected tissue.

## CONCLUSION

The percentage of apparently healthy udders reached 92.25 %. However, the percentage of quarters with high total bacterial count was 82 % reflecting the great need for stringent mastitis control policies especially identification and segregation of diseased buffaloes. The prevalence of environmental bacteria (isolates of Staphylococcus, Bacillus and Coliform, Table 2) points at the wide-spread bacterial contamination in the animal's environment. The histopathological alterations observed in the mammary tissue due to infection with mastitis pathogens resulted in decreased milk synthesizing activity caused by the replacement of the secretory tissues with the interalveolar stroma. The present data show that the rate of intramammary infection could be controlled through stringent mastitis management practices for lactating buffaloes, that may help to decrease cross-contamination. Moreover, segregation and proper treatment of infected animals and early culling of buffaloes showing any distortion in the mammary gland which may predispose the mammary tissue vulnerable to the mastitis pathogens may be a good help for having a mastitis-free-buffalo herd.

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## مدى انتشار الميكروبات المسبّبة لالتهاب الضرع والتغيرات الهستوباثولوجية فى الغـــدة اللبنية للجاموس المصرى

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لتقييم الحالة الصحية للغدة اللبنية فى الجاموس المصرى تم استخدام الفحص المظـــهرى والعزل البكتيرى للمسببات المرضية بالإضافة إلى دراسة هستولوجية لأنسجة الغدة. وقد أجـــرى فحص الغدد اللبنية مظهريا لعدد ٤٠٠ جاموسة. وقد تم إجراء اختبار العد الكلى للبكتريـــا وكـــذا تصنيف البكتريا المعدية وغير المعدية والمسببة لمرض التهاب الضرع فى عدد ٥٠٨عينة لبن تـــم جمعها من عدد ١٢٧ جاموسه. ووجد أن نسبة الغدد اللبنية غير الطبيعية تصل إلى ٧,٧٥ وكـــانت أكثر العيوب ظهوراً هى : الزيادة فى عدد الحلمات ثم الأرباع بدون فتحـــة حلمـــة والحلمـــة ذات الفتحتين ثم الضرع الجلدى والمشوه. واتضح أن ما يقرب من ٨٢% من العينات وجد بـــها عـــد بكتيرى كلى أكثر من المسموح به مما يشير إلى عدم توافر الظروف الصحية المناسبة ولقد كـــانت أكثر أنواع المسببات المرضيـــة هـــو المكـــور العنقـــودى (٤٨,١٨%) والميكروبـــات الســـبحية (٢٣,٦٩%) ثم الميكروبات العصوية (٩,٢%) يليها بكتريا القولون (٥,٦%).

وعند إجراء الدراسة الهستولوجية اتضح أن هناك تدهور شديد بالأنسجة المفرزة والتـــى تحولت إلى أنسجة ليفية ضامة مع انتشار واضح للخلايا الالتهابية والليفية وقد وجدت أيضا بعض الأجسام الدموية المتكونة فى فراغ بعض الحويصلات الضامرة. وكانت هذه التغيرات الهستولوجية مرتبطة بوجود أكثر من مسبب مرضى فى الفترة اللبنية.

ومما سبق يتضح أن هذه الحيوانات تحتاج إلى رعاية خاصة ومكثفة لتشـــخيص وعـــزل وعلاج الحيوانات المصابة ومن ناحية أخرى فإن استبعاد الحيوانات ذات الضرع المشوه ســـوف يكون له تأثيراً واضحا على تقليل فرص العدوى لأنسجة الضرع ذات الحساسية العالية للعـــدوى بالمسببات المرضية.